Fatigue Fractures

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■ *Fatigue (or stress) fracture of bone in military recruits has been recognized for many years. Most often it is a metatarsal bone that is involved but the tarsal bones, calcaneus, tibia, fibula, femur, and pelvis are occasionally affected. Reports of such fractures in the ribs, ulna and vertebral bodies may be found in the literature.*

In recent years, there has been increasing awareness of the occurrence of fatigue fractures in the civilian population. Weekend sportsmen, athletes in an early phase of training, and persons engaged in unaccustomed, repetitive, vigorous activity are potential victims of such a fracture.

The signs and symptoms, roentgenographic findings, treatment and etiology of fatigue fractures are dealt with in this presentation.

PAINFUL, SWOLLEN FEET in soldiers after long marches were first described in 1855 by Breithaupt, a German military surgeon.[6] He believed this condition was a traumatic inflammatory reaction in tendon sheaths and named it *Fussgeschwulst.*

Weisbach (1877)[14] believed that the lesion was in the ligaments and coined the term syndesmitis metatarsea. Pauzat (1887)[11] also pointed out its occurrence in soldiers and noted that there was palpable periosteal proliferation on the second, third or fourth metatarsals, the second being most common.

After these reports, other observers suggested various explanations of this lesion. In 1897, Stechow,[12] of the Prussian Guard in Madrid, using the newly discovered roentgen ray, defined the nature of the disorder. He reported on the roentgen examination of 36 patients and found that metatarsal fracture was the basic lesion. Many reports then followed; some investigators held that a fracture always occurred, while others noted that in a certain number of cases it did not develop. The descriptions of these lesions are now easily recognized to be of fatigue (march, stress) fractures.

It was gradually recognized that such fractures occurred in other bones. In 1905, Blecher[3] first reported on a case involving the femoral neck. Aleman (1929)[1] described a similar lesion in the tibia and Burrows (1948, 1940)[7,8] discussed cases of fatigue fracture in the fibula. Hullinger (1944)[9] reported on 53 cases of fractures of the calcaneus. In 1966, the author[4] reported on a series of femoral neck fractures which occurred in recruits undergoing basic training. Involvement in other areas, such as the ulna, humerus and ribs, has also been reported occasionally.

Relative frequency of involvement of various affected bones was obtained from a review of 700

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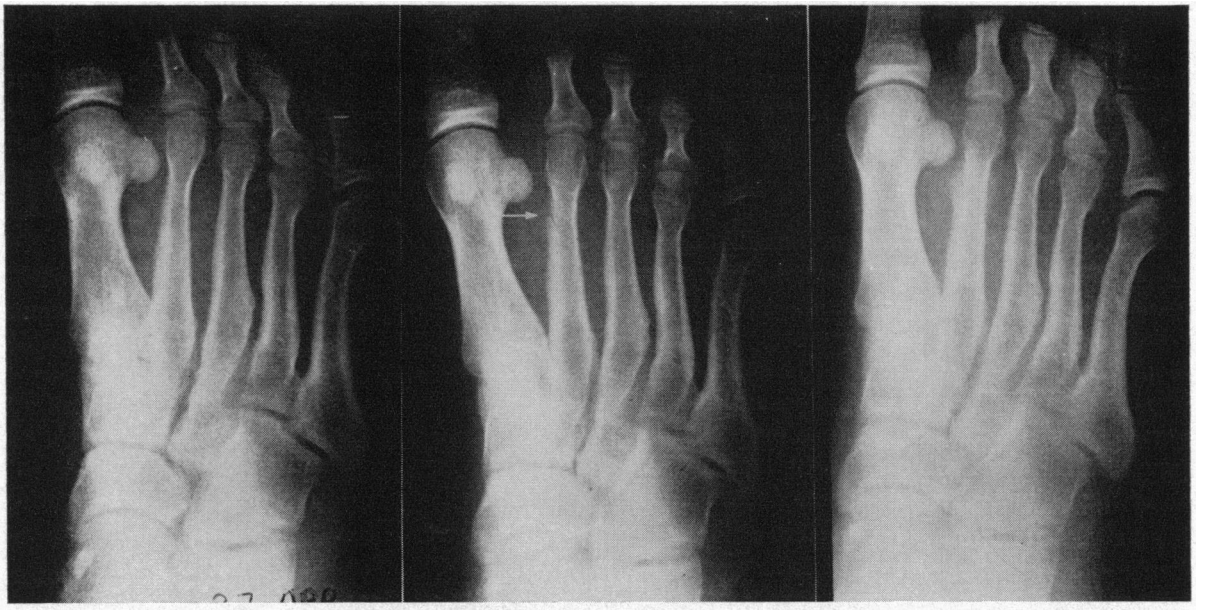

Figure 1.—Right forefoot of a 23-year-old recruit. Physical examination, approximately one week after onset of symptoms, revealed tenderness and swelling over the neck of the second metatarsal. Reading from left to right are: Initial x-ray film; ten days later, showing cortical crack (arrow) in the characteristic position in the medial cortex of the neck of the metatarsal; five weeks after onset of symptoms, showing callus formation.

cases of fatigue fracture which occurred during a three-year period at the Fort Ord Infantry Training Center. The incidence, expressed in per cent, was: Metatarsals, 51; calcaneus, 26; tibia, 17; femur, 4; fibula, 1; miscellaneous, including ischiopubic ramus, 1.

In the past, the lack of recognition of the true nature of the causative factors has led to a host of descriptive terms for this disorder. These include: swollen foot (Fussgeschwulst), syndesmitis metatarsea, march fracture, Deutschländer's disease, pied forcé, insufficiency fracture, overload fracture, wear-and-tear fracture, recruit's disease, periostitis ab exercitio, osteopathia itineraria, soldier's fracture, spontaneous fracture, pseudofracture, insidious fracture, creeping fracture, and stress fracture. Fatigue fracture is now considered to be the best descriptive term for this condition.

General Considerations

Fatigue fracture is a term used to describe three distinct but overlapping responses of normal bone to repeated minor stresses. A partial or complete fracture through the affected bone is the most obvious and well known response. The second is a microscopic fracture of bone which can be inferred to have taken place because of the subsequent callus formation or the later clinical progress of the disorder. The third response, a fatigue, or stress, reaction, is now being recognized with increasing frequency. In this condition, the affected area of the bone becomes osteoporotic; later, there is progressive development of endosteal and periosteal callus in response to continued activity. Apparently, timely curtailment of the inciting activity precludes the development of a true fracture.

Spontaneous fracture is a more general term which should not be used in referring to fatigue fracture, because a spontaneous fracture may occur either as a result of stress or fatigue of bone or in conjunction with pathologic disorders of bone. In a fatigue fracture, there is no evidence of systemic or local disease associated with any pathologic condition of the bone.

The general characteristics of fatigue fractures were described by Burrows in 1940:[8]

> Generally, involvement of the shaft of a long bone;
> Onset without violence;
> Frequently, association with prolonged muscle effort;
> Absence of any audible snap or of any suspicion by the patient that the fracture has occurred;
> Usually pain as the outstanding symptom;
> Liberal formation of callus and its subsequent development into mature bone;
> Presence of a variable degree of edema.

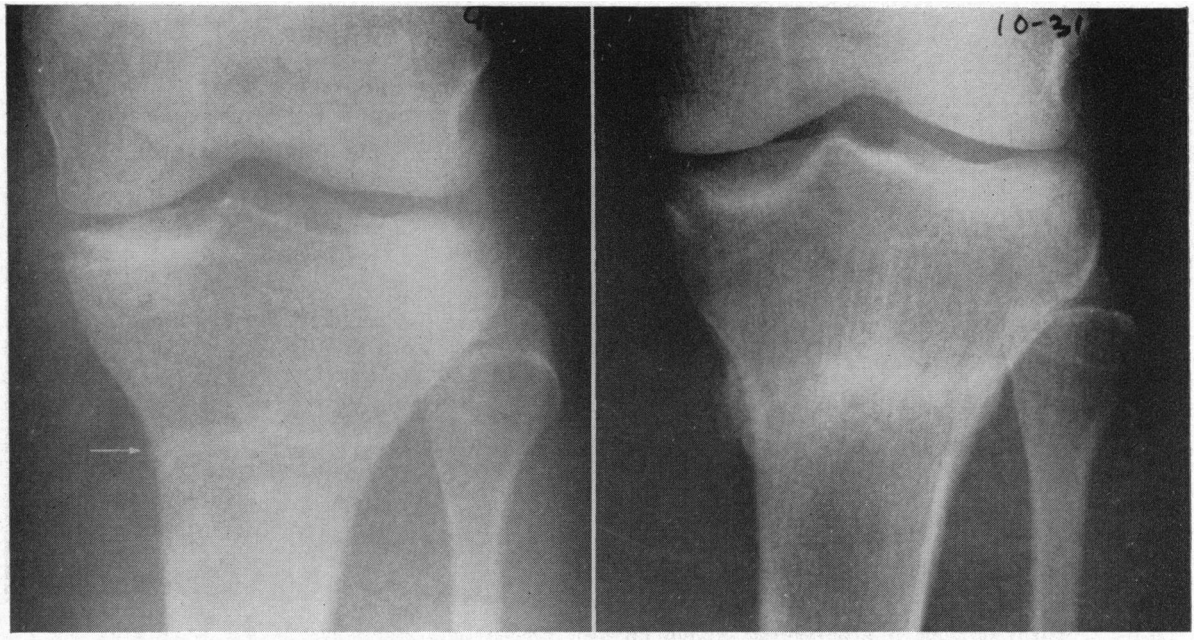

Figure 2.—Left knee of a 23-year-old recruit. At time of examination, pain had been present for two and one-half weeks. There was tenderness over the proximal tibia below the knee joint. At left is initial x-ray film and at right a film taken five weeks later when the patient was asymptomatic.

Whenever a possible diagnosis of fatigue fracture is encountered the quality of the bone may be questioned. Although the affected bone may have inert defects in crystalline structure, trabecular orientation or ground substance, we cannot detect, by the gross clinical methods of evaluation now available, any abnormality of bone affected by fatigue fracture.

Symptoms and Signs

The one constant symptom in this disorder is pain associated with physical stress of the part. Generally, there is associated swelling and tenderness over the involved part of the bone. Occasionally, ecchymosis or erythema may be seen. The pain and swelling usually subside with rest of the affected part. These symptoms and signs are, of course, most frequently encountered in persons undergoing basic military training, but they also occur in weekend sportsmen or in athletes performing at their maximum. These fractures, previously thought to be almost exclusively a military problem, are being recognized more and more frequently in civilian practice. Among the most important factors in accurate diagnosis are a history of pain associated with generally unaccustomed stress, an awareness by the physician of the possibility of the disorder and the availability of serial roentgenograms to confirm the suspicion.

Roentgenograms

Although the diagnosis of fatigue fractures may be tentatively established by an appropriate history combined with findings of localized pain, tenderness and edema over the affected bone, confirmation depends upon the progressive changes of the disorder as observed roentgenographically.

There are probably many fatigue reactions in bone which, owing to reduction of the initiating stress, do not reach a point at which signs are roentgenographically diagnostic. This possibility may explain the so-called "growing pains," "shin splints" and "ligamentous strains," which are poorly understood.

Early films are generally normal. Frequently, changes are not observed for 14 to 21 days or longer. Callus (either endosteal or periosteal) or a cortical crack may then be seen. The rate and degree of progression depends on the amount of continued stress and on the particular bone or area involved. In the metatarsal or fibula, a crack may be the first roentgenographic sign. In the tibial plateau, femur, or femoral neck, endosteal or periosteal callus may be the first sign observed on x-rays. Complete fracture, if it develops, generally occurs two to three weeks after the onset of symptoms. Figures 1 to 6 illustrate the development of these fractures in various bones.

It should be pointed out that, because of the length of time between the onset of symptoms and the development of findings that are visible on x-ray films, many cases of fatigue fracture are completely missed or misdiagnosed, especially in civilian practice (since the symptoms subside with decrease of activity, serial x-ray films are not obtained).

Differential Diagnosis

Occasionally the diagnosis of fatigue fracture may be quite difficult and, at times, must be established largely by exclusion. Several conditions may be confused with fatigue fracture and must be distinguished. These include:

- Pathologic fractures seen in congenital defects, general systemic disease and metastatic bone tumors;
- Pseudofractures, or "Umbauzonen," such as occur in Paget's disease, osteomalacia, rickets and Milkman's syndrome;
- Primary malignant disease of bone, such as osteogenic sarcoma, Ewing's tumor or reticulum cell sarcoma;
- Osteitis due to tuberculosis or syphilis;
- Simple fracture due to violence;
- Osteomyelitis.

Pathologic fractures and, especially, so-called pseudofractures are the cause of most of the problems in differential diagnosis.

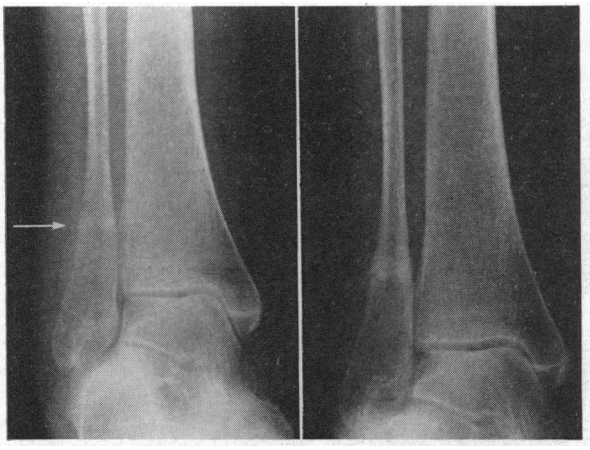

Figure 3.—Right ankle of a 68-year-old woman. Pain had been present for two and a half weeks and there was edema and tenderness over the distal fibula above the ankle joint. In initial x-ray film (left) fatigue fracture (arrow) is seen at upper border of syndesmosis. Treatment included a Gelocast wrapping and limited weight-bearing. Film taken five weeks later (right) shows progression of healing.

In general, pathologic fractures can be distinguished because they occur in obviously diseased bone affected by such disorders as tumor, leukemia, osteoporosis and metabolic bone disease.

Pseudofractures may be described as transverse zones of rarefaction varying in width from less than a millimeter to more than a centimeter, and usually symmetrical in distribution. In addition to their occurrence in rickets, osteomalacia and Milkman's syndrome, they have been described in association with renal rickets, coeliac disease, chronic idiopathic steatorrhea and Paget's disease. They are usually of two types. The first type is associated with malacic diseases and appears as small subperiosteal notches in the cortex when viewed tangentially, or as small, irregular, circular, punched-out zones of decalcification when viewed *en face*. This type slowly progresses across part or all of the bone as a band of decalcification. Periosteal reaction, sometimes observed in curved bones, may be apparent only on the concave side. The second type of pseudofracture is not associated with malacic disease and appears as cracks or fissures extending through the cortex on one side, most often on the convex surface of pathologically curved bones. Defects of this type are seen in Paget's disease.

These defects, or "pseudofractures," differ from actual fractures in several respects: They develop spontaneously without gross trauma and there is no crepitus or undue mobility except for a slight degree of "elastic give." The similarity of "pseudofracture" to fatigue fracture and the source of confusion in diagnosis are apparent. However, as now accepted, they are two entirely separate entities. The importance of accurate differentiation for purposes of treatment and prognosis is obvious.

Fatigue Fracture Following Immobilization. I have recently seen a type of fracture that I feel deserves mention because it might at some time also perplex others. This fracture is associated with increased activity or weight-bearing after a period of bed rest or immobilization. It has elements of both a true fatigue fracture and a pathologic fracture in that it is seen in bone which has become osteoporotic because of disuse.

Treatment

Early recognition of a fatigue fracture is of primary importance in treatment. This is especially true in the case of fractures of the femoral neck,

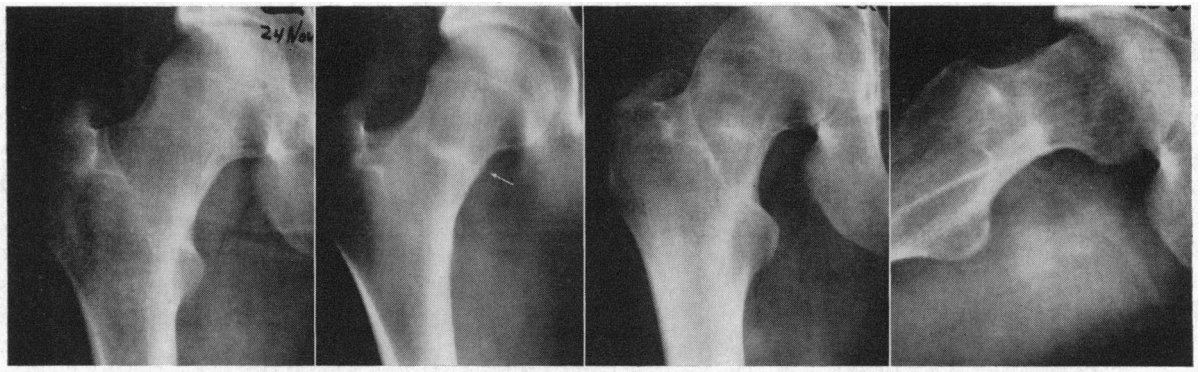

Figure 4.—Right hip of a 24-year-old recruit with a history of pain in the right hip which had lasted approximately ten days. Reading from left to right are: Initial x-ray film, interpreted as normal; tomogram of the hip three and a half weeks later, demonstrating fatigue fracture of the femoral neck (arrow) with endosteal and slight periosteal callus; anteroposterior and lateral views nine days later when the patient was asymptomatic.

where displacement may lead to serious or even disastrous consequences. Treatment for any bone in an early stage of the lesion may consist simply of curtailment of activity or rest of the part. When x-ray studies suggest that fracture may occur, or that a complete fracture already has occurred, immobilization in a cast is usually indicated. (Decision as to whether to apply a cast depends on the severity of the symptoms and the particular bone involved.) In the case of fractures of the femoral shaft and, particularly, the femoral neck, open reduction and internal fixation may be required.

Etiology

In the past, many theories regarding the cause of fatigue fractures have been advanced. These include: Nutritional deficiency; polyhypovitaminosis; local or systemic infection; vascular changes; pull of muscles and ligaments; and[5] "disproportion" between "required" and "inherent" capacity of the bone to bear stress. Most of these theories have been discarded, in part or in whole, in the light of more recent information.

Since the relationshp of fatigue, or stress, fracture to repeated minor stresses (rather than to one specific injury) has been recognized, frequent efforts have been made to equate this phenomenon to fatigue disruption in metal and other inert material. This correlation contributes somewhat to our understanding of etiologic factors, but the living, dynamic quality of the bone in which the fracture occurs must be emphasized. It is common knowledge that bone is remarkably sensitive to and indicative of the amount of stress or use to which it is subjected. That disuse or immobilization leads to osteoclastic resorption is well known. It is, perhaps, more surprising that overuse also leads initially to resorption of bone. Weinmann and Sicher[13]

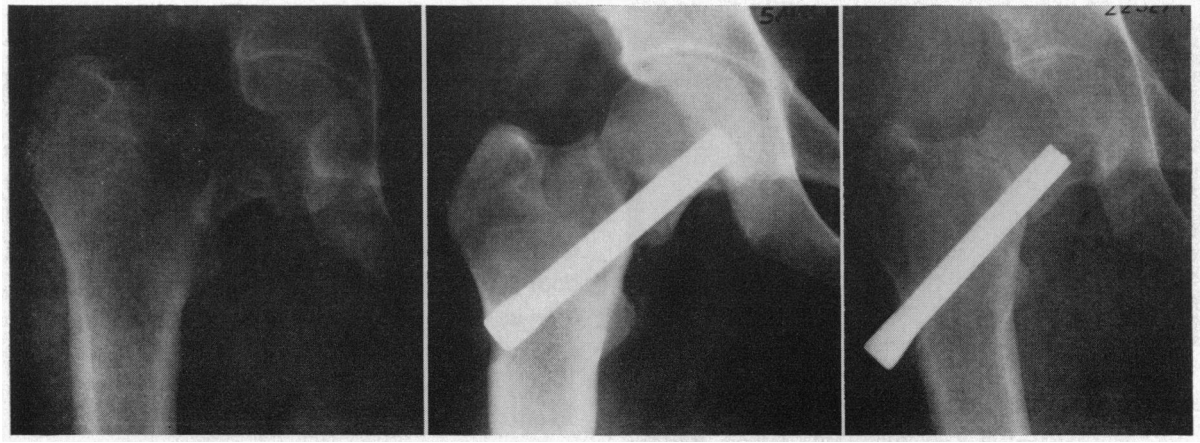

Figure 5.—Right hip of a 24-year-old recruit who had had pain in the groin and thigh for two weeks. During marching, the right leg suddenly gave way. At left is initial x-ray film, anteroposterior view. Center panel, five days later, after Smith-Petersen nailing. Despite poor reduction, the fracture eventually healed. Film at right was taken six months after nailing. The nail was removed one year after insertion.

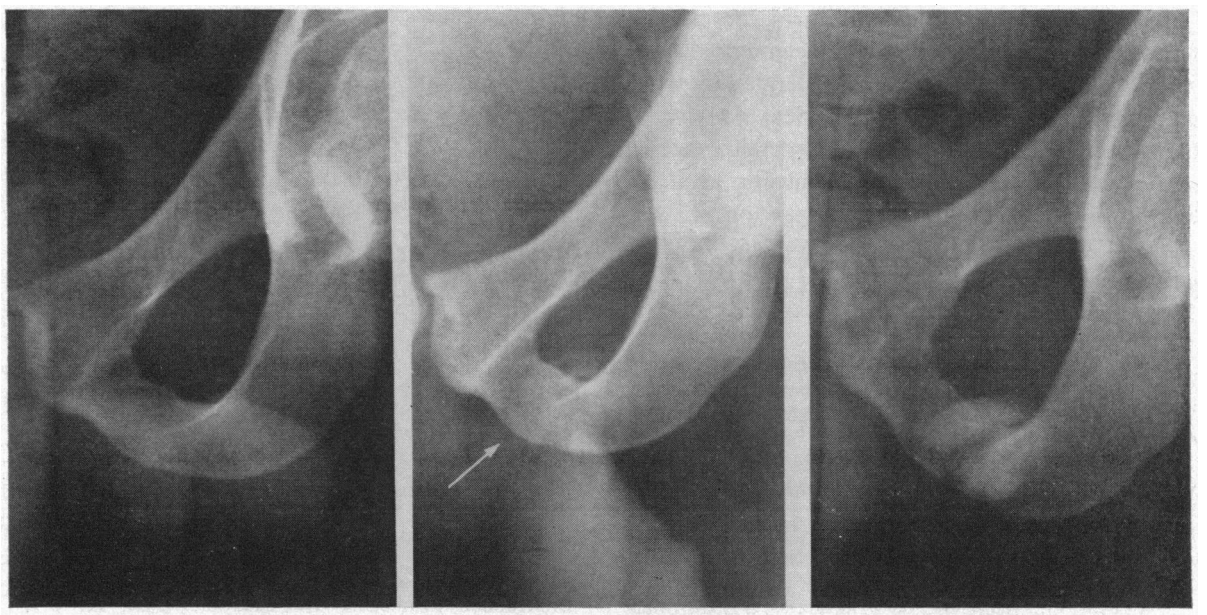

Figure 6.—Typical progression of a fatigue fracture of the ischiopubic ramus. X-ray films were taken one and a half weeks, three and a half weeks and eight weeks after onset of symptoms. Initial film appears normal; progressive formation of callus is seen in second and third.

said, "Increase of pressure or tension beyond the limits of tolerance leads to destruction of bone by resorption. . . ."

Johnson and coworkers[10] studied, with use of specimens taken for biopsy, the development of fatigue fracture of the tibia in 30 cases in which the duration of symptoms and roentgenographic signs ranged from seven days to seven months.

During the first week, localized pain, tenderness, swelling and erythema were associated with very active osteoclastic resorption of the cortex as shown by local osteoporosis. Before resorption began, lamellar birefringence in polarized light was reduced. The normal sharply structured lamellar pattern became smudged or lost in the area of fracture. There appeared to be an "unraveling" of marginal lamellar fibrils with consequent decrease in parallel orientation of the fibrils in the collagen bundles that constitute the lamellae. Resorption followed the partial disorganization of the collagen lamellar structure. During the second week, periosteal (occasionally endosteal) callus formation occurred. Resorption was complete at three weeks and callus formation was maximal at approximately six weeks.

Remodeling, or transformation, from circumferential lamellar bone to osteonal bone occurs between 18 and 28 years of age. All biopsy specimens showed development of the lesion within unosteonized lamellar bone. It appears, therefore, that fatigue fracture may represent an acceleration of the normal internal remodeling of circumferential lamellar bone to adult osteonized bone.

Knowledge of the mechanisms by which stress affects osteogenesis and the replacement, or turnover, of bone is, therefore, necessary before complete understanding of the causes of fatigue fractures is possible. Recent work by Bassett[2] on electrical effects in bone appears to suggest a link between mechanical stress and biological activity in bone. Because bone is so highly crystalline, bending forces on bone generate electricity, roughly in proportion to the amount of its deformation. This is due to a piezoelectrical effect produced by one or all of three mechanisms: (1) Stress or bending of collagen fibers; (2) bending of mucopolysaccharide molecules; (3) stress on collagen-hydroxyapatite interface (a semiconductor of the P-n type). Areas under compression (concave) are negatively charged, while convex areas are positively charged. Deposition of bone is influenced by weak, artificially induced currents and occurs in the area of electronegativity, or concavity. The converse, that is, that electropositivity causes osteoporosis of bone, has not been demonstrated.

Present etiologic knowledge of fatigue fractures can be summarized as follows. Fatigue fracture begins with excessive elastic deformation and recoil of bone, causing an "unraveling" of the lamellar structure of a segment of older circumferential

bone. Subsequent local resorption of the circumferential bone is followed by replacement with osteonal bone, a normal process accelerated by stress and, perhaps, mediated by electrical activity. If this acceleration is too rapid, resorption occurs more rapidly than replacement, causing local osteoporosis followed by callus formation. If mechanical stress is too great, complete fracture occurs.

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